THE TAMILNADU DR. M G R MEDICAL UNIVERSITY


*A Dissertation on*

**"EVALUATION OF PROGNOSTIC FACTORS IN TRAUMATIC ACUTE SUBDURAL HEMATOMA"**

***submitted in partial fulfillment of requirements***
***for M.Ch. Branch II***
***(Neurosurgery- Five years Course) Examination***
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**INSTITUTE OF NEUROLOGY**
**MADRAS MEDICAL COLLEGE**
**CHENNAI**

# CERTIFICATE

Certified that the Dissertation titled "**EVALUATION OF PROGNOSTIC FACTORS IN TRAUMATIC ACUTE SUBDURAL HEMATOMA"** is a *bonafide* work done by **DR. K.R. RAJAVADIVEL** during the period of his M.Ch. (Neurosurgery) Course from June, 2004 to February, 2009.

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# INTRODUCTION

Head injury is a most severe form of injury occurs by road traffic accident and fall. Acute subdural hematoma (ASDH) is a fairly common finding in patients admitted in unconscious state following trauma. This condition causes a very high mortality even in best centers ranging from 50% to 80%. The causes of mortality may be due to various factors including co morbid factors, severity of associated brain injury etc. There have been many studies analyzing the impact of various factors affecting the prognosis in acute subdural hematoma factors like the age, conscious level , brain stem reflexes, computerized tomography (CT) findings, the time of injury to time of surgical intervention have been found to be important in deciding the outcome in acute subdural hematoma.

# AIM OF STUDY

This study aims at analyzing the various factors which affect the outcome in acute traumatic subdural hematoma in local population.

# REVIEW OF LITERATURE

Acute subdural hematoma is collection of blood in the subdural space associated with or without contusion, intra cerebral hemorrhage and subarachnoid hemorrhage with the clinical presentation between 0 to 48 hours. Sub acute subdural hematoma implies clinical presentation above 48 hours after the time of injury. Chronic subdural hematoma implies clinical presentation more than 3 weeks of injury.

The mode of injury ranging from road traffic accident, train traffic accident, fall from height, assault, disasters like cyclone injury due to inertial forces causing impact energy which makes the brain to accelerate and decelerate with in the skull. This strain occurred is confined to brain surface causing injury to brain surface and bridging veins. If the duration of acceleration is prolonged the strain penetrate the deeper in to the brain causing diffuse axonal injury. Another source of SDH caused by injury involving laceration of brain or rupture of arterial and venous structure. Injuries may be coup injury where SDH pertains to area below the injury sites (frontal, temporal, parietal, and occipital region) contre coup injuries pertains to injury that occurs at the opposite site of impact injury. Some hypothesis state that the acute subdural hematoma (ASDH) is

related to cerebral vertex displacement and diffuse axonal injury (DAI) to local Green-Lagrange strain. Worst-case of bridging vein strains are produced on the contre-coup side . Given that axons in the corpus callosum are highly oriented, Green-Lagrange strain was resolved in the fibre direction. It is found to be close to the minimum principal strain, indicating a degree of natural, teleological protection for the axons.

## AETIO PATHOGENESIS

Acute subdural hematoma is seldom an isolated lesion. These are patients in whom the extensive associated contusion and laceration may be the dominant feature and SDH only a relatively unimportant co existant pathological finding. A thin layer of subdural blood may commonly seen in association with traumatic intra cerebral hematoma which from a pathological stand point was difficult to differentiate from cerebral contusion with hemorrhage. Some times SDH can be divided in to simple SDH, SDH with contusion brain, SDH with intra cerebral hematoma. Based on aetio -pathogenesis Miller and Statham classified (1995) acute subdural hematoma in to three types (1) Hematoma associated with laceration of the brain usually at temporal pole in which there is a mixture of intra cerebral contusion and hemorrhage and an acute subdural hematoma a condition known burst temporal lobe. (2)

Acute subdural hematoma that results from tearing of a bridging vein between the surface of the brain and one of main venous sinuses. (3) Hematoma due to bleeding from a small artery on the surface of the brain that there have been injuries by a overlying fracture of skull. The patho physiological evidence support the belief that in a ASDH the extent of primary underlying brain injury is more important than subdural clot itself indicating the outcome, Which implies mortality and morbidity related to the extent of underlying brain damage rather than the extent of subdural clot. In vast majority of cases aetio-pathogenesis clinical picture and prognosis cannot be explained simply on the basis of a surface clot producing focal compression of brain.

## ACUTE SUBDURAL HEMATOMA AND BRAIN SWELLING

While cerebral contusion, laceration or intra cerebral hemorrhage are obvious on CT scan and can be seen with naked eye at operation or at autopsy. Diffuse axonal injury which may occasionally be supported on the basis of CT finding can only be observed in carefully conducted pathological studies. Acute subdural hematoma may be an epiphenomena of a primary impact lesion of variable severity that is a diffuse axonal injury. It is there fore understandable that the patient with ASDH who remains unconscious from the moment of head injury as may do

clinically reflect the severity of this primary damage rather than the compressive effect of any associated SDH.

## ACUTE SUBDURAL HEMATOMA AND ISCHEMIC NEURAL DAMAGE

Ischemic brain damage is the commonest neuro-pathological abnormality found in patients who die of acute subdural hematoma. Angiographic evidence of cerebral vasospasm in patient with severe head injury is a cause of brain ischemia. While the precise mechanism by which ASDH causes underlying brain ischemia remains uncertain, there is evidence to suggest that neither focal nor generalized intracranial pressure could adequately explain the condition. The possibility of direct effect of some vaso active substances released by the blood clot being responsible for the ischemia seems attractive in micro dialysis techniques studies shown acute subdural hematoma was associated with massive release of glutamate, to 6 times the normal level, for 20 minutes in the underlying cortex. There is profound increase in level of extra cellular exicitatory amino acid in the ischemic zone under the SDH. It is postulated that the EAA induced increase in local cerebral glucose utilization result in a break down of the metabolism and cerebral blood flow coupling. There is production of ischemic brain damage associated with tissue hyper metabolism in SDH following use of glutamate

antagonist. The compression of micro circulation could be responsible for the ischemia. Patients with undamaged arachnoid barrier to be due to the shielding of the cortex from the neuro toxic and vaso active substances preventing the ischemic and edemo genic response in the brain tissue. It is known that such vaso active and neuro toxic substances are released not only by the subarachnoid and subdural blood but also by the brain damaged by an impact injury or ischemia induced by hypo perfusion and hypotension occurring at the time of injury.

## ASDH AND BRAIN INJURY, ROLE OF SPET

This investigation examined the role of brain perfusion single-photon emission tomography in traumatic head injury patients. In patients investigated by SPET with technetium-99m-hexamethyl propylene amine oxime (Tc-HMPAO) revealed CT/MRI-negative abnormalities, such as hypo perfusion in the contre-coup region, frontal hypo perfusion related to personality change, cerebellar hypo perfusion associated vertigo ,Brain stem axonal injury and cortical axonal injury leading to post traumatic amnesia in temporal lobe lesions. Frequently, SPET may be the only examination to reveal perfusion abnormalities which are related to symptoms in the absence of other objective findings.

## ACUTE SUBDURAL HEMATOMA AND BRAIN SWELLING:

Zumkeller et al, recorded a mortality of 50% when midline shift exceeded the thickness of hematoma by 3 mm in the CT scan. How ever if the midline shift exceeded hematoma thickness by 5mm, the survival rate was only 25% at surgery itself and the brain herniates through the craniotomy, following evacuation of the clot, making dural closure impossible. In some times a post-operative CT scan shows cerebral hemispheric enlargement, with persistent or even increased midline shift absence of any residual hematoma. This result in 70 to 80% mortality .Brain edema in fatal head injury patients is a common cause of brain swelling. In case of fatal head injury more recent studies established the high incidence of impaired cerebral blood flow, decoupling of cerebral blood flow and metabolism and failure of auto regulation resulting in cerebral ischemia and infarction with consequent brain edema. Excito toxic substances like glutamate and aspartate, free radicals, and platelet activating factor have been implicated. A cascade of events initiated by the primary traumatic event is complicated by secondary events of local and generalized increase in intracranial pressure, abnormalities of cerebral perfusion and ischemia.

**CLINICAL FINDING:**

Rapidly evolving symptoms of drowsiness to unconscious, disorientation to no verbal response and obeying commands to no motor response are the presenting symptoms. Transtentorial herniation is indicated by bradycardia associated with hypertension (cushing response). Localizing signs includes ipsilateral pupillary dilatation with impaired reaction and motor deficit contralateraly . False localising signs include contralateral pupillary dilatation due to opposite optic nerve injury. Kernohan notch is motor weakness of same side due to compression of the contra lateral cerebral peduncle against tentorial edge. Dolls eye movement is absent in cases of brainstem compression due to uncal herniation caused by massive ASDH and shift of cerebral hemisphere.Posttraumatic seizure may occur in cases of ASDH associated with contusion which may be fatal.

## PROGNOSTIC FACTORS

### 1. AGE

Age is a strong independent factor for prognosis in management of acute head injury. Patients in old age group and infants has poor outcome. Patients in childhood and in middle age group survival rate is higher. Patients with thin build has poor outcome. A study by Tallon et al (2008) regarding the epidemiology of surgically treated acute subdural and epidural hematomas in patients with head injuries, a population based study showed median age of injury was 45 years and 80% of the cohort were male.

## 2. GLASGOW COMA SCALE

The clinical level of consciousness at the time of admission is noted by Glasgow coma scale.

It includes clinical findings of eye opening, motor response and verbal response.

| EYE OPENING | SCORE |
|---|---|
| Spontaneous eye opening | 4 |
| Eye opening to verbal response | 3 |
| Eye opening to painful stimuli | 2 |
| No eye opening to painful stimuli | 1 |

| MOTOR RESPONSE | SCORE |
|---|---|
| Obeys command | 6 |
| Localizes to painful stimuli | 5 |
| Flexion withdrawal response to painful stimuli | 4 |
| Abnormal flexion response to painful stimuli | 3 |
| Extension response to painful stimuli | 2 |
| No response to painful stimuli | 1 |

| VERBAL RESPONSE | SCORE |
|---|---|
| Oriented | 5 |
| Disoriented | 4 |
| Inappropriate words | 3 |
| Incomprehensible sound | 2 |
| No verbal response | 1 |

The severity of injury is noted by the GCS as follows,

| GCS | SEVERITY |
|---|---|
| 3-8 | SEVERE |
| 9-12 | MODERATE |
| 13-15 | MILD |

GCS with 3-8 patients show very poor outcome.

GCS with 9-12 patients show moderately better outcome.

GCS with 13-15 patients show good outcome scale.

## 3. PUPILLAY RESPONSE AND OCULO CEPHALIC RESPONSE (OCR):

Patients with pupils bilaterally reacting to light with normal size and positive oculocephalic response has good outcome. Patients with pupils bilaterally 3mm in diameter and sluggishly reacting to light with normal or impaired OCR has moderately better outcome. Patients with pupils bilaterally 4mm not reacting to light and absent OCR has poor outcome. A fixed pupil is defined as exhibiting no constrictor response to bright light.

## 4. CT SCAN FINDING:

The classic CT appearance of acute SDH is a crescent shaped homogeneously hyper dense extra axial collection that spreads diffusely over the affected hemisphere concavely. 40% of acute SDH have mixed hyper and hypo dense areas that reflect un clotted blood. Serum extruded during clot retraction with in the subdural hematoma is due to arachnoid laceration. Associated finding of mass effect, compression of ipsilateral ventricle and in more severe cases with midline shift and obliteration basal cisterns. More than 5mm thickness of hematoma is accountable. In acute subdural hematoma 60% is hyper dense and 40% is mixed hyper and hypo dense. Attenuation value is 65 to 95 HU. Swirl sign implies lower density un clotted whole blood swirls in to a region of clotted blood that is hyper dense, extra axial blood without a skull fracture and an extra axial collection of blood that crosses sutures but is limited by the falx and tentorium. Duret hemorrhages are delayed secondary brain stem hemorrhages seen in CT brain located in the ventral and para median aspect of upper brain stem (Mesencephalon and pons). Patients with severe midline shift and basal cistern effacement has poor outcome. Patients with CT finding without midline shift and normal basal cisterns have good outcome.

## 5. TIME BETWEEN INJURY AND SURGICAL INTERVENTION

Early transport to trauma center and resuscitation and shifting for emergency surgery needs to be very quick in reducing the duration of time interval and better outcome. Several data showed surgical intervention before 4 hours of injury resulted in better outcome.

## MANAGEMENT OF ACUTE SUBDURAL HEMORRHAGE

The management of acute subdural hemorrhage begins immediately at the scene of road traffic accident with basic and advanced interventions performed by pre hospital personnel and is strongly presumed to have a subsequent distinct impact on outcome. The basic management of airway, breathing and circulation in the pre-hospital phase of care has been shown in other studies to correlate with improved outcome. As soon as patient is transferred to trauma -ward clinical finding of GCS, pupillary response, vital signs should be recorded and intravenous fluid, antibiotic, analgesics and control of bleeding from the lacerated wound by suturing should be done. Immediate CT scan of the patient should be taken. Posttraumatic seizures can be managed by intravenous antiepileptic (phenytoin, sodium valporate).

## INDICATION FOR SURGERY

Thickness of subdural hematoma more than 10mm and mid line shift of more than 5mm on computed tomography should be surgically evacuated regardless of the patient Glasgow coma scale. A comatose patient (GCS less than 9) with an subdural hematoma less than 10mm thickness and a midline shift of less than 5mm on computed tomography should undergo surgical evacuation ASDH. If the GCS SCORE had decreased between the time of injury and hospital admission by 2 or more points they should be operated immediately. Patients who present with asymmetrical dilated or fixed dilated pupils and if the ICP exceeds 20mm hg they should be operated immediately.

## SURGICAL MANAGEMENT

In ASDH and associated contusion a large frontal temporal parietal craniotomy was done in emergency operation theatre with patient in supine position, head tilted to the opposite side of ASDH , under general anesthesia a trauma flap made over frontal, temporal, parietal region on the side of ASDH. Scalp flap elevated towards the temporal base. Six burr holes were laid over frontal temporal and parietal region and craniotomy bone flap raised. Temporal base bone was nibbled to prevent uncal herniation. Dura was opened in a stellate shape to prevent brain swelling. Exposure of frontal and temporal poles and also along the sagittal sinus is especially important by opening the Dura generously and clot was removed by suctioning. Bleeding points on the cortical surface were coagulated and diffuse oozing can be controlled with hemostatic agents such as thrombin soaked gelfoam or surgifoam. Contusion of the brain was removed by suctioning and electro coagulation. Dural closure was attempted in cases of no brain edema followed by removal of bone. Wound was closed in layers. Postoperative care in head injury ICU with ventilator support antibiotics, anti-edema drugs, anti-epileptics, analgesics were instituted. Physiotherapy and tracheostomy care given adequately. Death was more common in severely injured poly trauma patients.

**GLASGOW OUTCOME SCALE (GOS):**

GOS is commonly used to grade the outcome in head injury, which is as follows:

| SCORE | RATING | DEFINITION |
|---|---|---|
| 1 | Death | Patient did not survive |
| 2 | Persistent vegetative state | Minimal responsiveness |
| 3 | Severe disability | Conscious but disabled depend for daily support |
| 4 | Moderate disability | Disabled but independent |
| 5 | Good recovery | Resumption of normal life |

There were several studies regarding the outcome of patients with traumatic acute subdural hematoma.

Klun et al (1984) studied the factors influencing the outcome in acute subdural hematoma in a review of 330 cases to analyse the clinical signs which influence the outcome such as age, pupillary changes, dynamics of the clinical development and the state of consciousness and finally some conclusions were made.

Athiappan et al (1993) studied the influence of basal cisterns, midline shift and the pathology on outcome in head injured patient. The state of the cisterns and midline shift was correlated with the type of intracranial pathology and Glasgow Coma Scale (GCS) scores. The state of the cisterns and midline shift was more important for those with single contusions and intra-cerebral haematoma (ICH) than for those with multiple lesions, extra dural haematoma, subdural haematoma, diffuse cerebral oedema and normal CT scan. The state of the above two parameters when correlated with GCS score, showed that they were important for those with higher GCS scores. This indicates that the status of the cisterns and midline shift is correlated with the type of pathology and GCS score rather than these parameters taken alone in for prediction.

Cruz et al (2001) studied Improving the clinical outcomes from acute subdural hematomas with emergency preoperative administration of high doses of mannitol and concluded that emergency preoperative HDM administration was associated with improved clinical outcomes for patients with acute subdural hematomas. Preoperative improvement of abnormal pupillary widening and better postoperative control of intracranial hypertension and associated relative cerebral hyper perfusion seemed to be relevant factors associated with improved outcomes.

Martynov IuS, et al (2000) In a Follow-up study of patients operated for traumatic subdural hematoma clinical manifestations were analyzed (8 main syndromes of the remote period) as well as the degree of neurologic rehabilitation and the level of social-occupational adaptation. Such adaptation appeared to be rather high: 66.1% of the patients were able to resume work. The highest lethality was in the acute period; the worst rehabilitation and follow-up adaptation were observed in elderly and old patients who were in comatose state before the operation and had severe accompanying contusion of the brain. Policy of drug treatment was determined in patients with traumatic subdural hematomas regarding peculiarities and manifestations of the syndromes (urgent operation, if necessary--cranioplasty, psychological support in the remote period, resorption therapy and symptomatic drugs).

Piotrowski WP et al (1995) retrospectively studied, the prognosis of acute subdural hematoma considered with reference to the data of 255 patients who underwent surgery because of acute subdural hematoma during a period of 10 years in the Department of Neurosurgery of the Landesnervenklinik in Salzburg. The mortality rate was 43.5%, and 21.6% had a full functional recovery. The findings of the study confirm that the preoperative neurological condition, the age, the kind of

accident sustained and the extent of concomitant brain injury influence on the prognosis decisively.

Phuenpathom et al (1993) studied Outcome and outcome prediction in acute subdural hematoma. This study is based on a series of 109 consecutive head injured patients with the CT scan diagnosis of acute subdural hematoma. The overall outcome was assessed at 6 months after injury using the Glasgow Outcome Scale. By logistic regression analysis a small set of clinical features (the best sum Glasgow Coma Scale score within 24 h after admission, and pupillary inequality) revealed as significant prognostic features. The method described allows bedside predictions in individual future cases.

There have been several studies on the impact of the various prognostic factors in ASDH.

Mizunom et al (1989) evaluated prognostic factors in acute subdural hematoma and the significance of serum Fibrinogen degradation products (FDP) concluded that a high serum FDP values were related to poor outcome.

Wilberger et al (1991) have analyzed 101 cases of ASDH. The overall mortality was 66%. They found that he following variables

statistically correlated (p less than 0.05) with outcome; motorcycle accident as a mechanism of injury, age over 65 years, admission GCS score of 3 or 4, and postoperative ICP greater than 45 mm Hg. The time from injury to operative evacuation of the acute subdural hematoma in regard to outcome morbidity and mortality was not statistically significant.

Kotwica & Brzeziński (1993) have analyzed 200 cases of ASDH. 63% of them were surgically treated within the first 4 hours after head injury; the others were operated on 4 to 16 hours after trauma. All patients had GCS below 10 for the whole time period from trauma to surgery. Younger patients 18-30 year old had lower mortality-25%, while patients above

50 revealed 75% mortality. Analysis of operative timing and outcome, no benefit revealed when surgery was performed within first 4 hours.

Dent dl et al (1995) evaluated 211 cases of ASDH out of which 128 cases were conservatively managed and 83 patients underwent craniotomy. The outcome predictors were GCS, injury severity scale,

ICP, direct admission to a trauma center, presence of SAH and the time of operation.

Massaro et al (1996) have studied 127 cases of operated ASDH, with mortality rate of 57%. They found that GCS and CT scan findings were found to be the most important prognostic variables. Timing of operative intervention for clot removal with regard to outcome was not statistically significant.

Cook et al (1996) in a Westmead head injury project report with a prospective study of 2 yr duration of 103 cases of ASDH concluded that age, hypoxia, hypotension, response to intracranial pressure control and two CT scan features, midline shift as measured from the septum pellucidum and cerebral edema, were all significant in predicting outcome. Time from injury to treatment, initial pupil response, lucid interval and compression of brainstem cisterns on CT scans statistically failed to predict outcome.

Koc et al (1997) have studied 113 cases of ASDH. The overall mortality was 60%. They concluded that age and associated intracranial lesions were related to outcome. Severity of injury and pupillary response were the most important factors for predicting outcome. Time from injury

to surgical evacuation and type of surgical intervention did not affect mortality.

Kayseri et al (1997) studied outcome and outcome predictors in ASDH. They concluded that the severity of injury and pupillary response were the most important factors in predicting the out come.

Yamaura et al (2001) studied outcome prediction in severe head injury; analyses of clinical prognostic factors retrospectively in 272 patients includes age, GCS, pupillary response, OCR and parenchymal lesions. They graded diffuse brain injury into 4 grades. Grade 1 included injuries with no visible pathology. Grade 2 included all injuries in which the cisterns were present with midline shift of less than 5mm. In Grade 3 cisterns were compressed or absent with midline shift of less than 5mm. Grade 4 included all injuries with a midline shift of more than 5mm.The mass lesions were categorized in to 3 subgroups EDH, ASDH, ICH. The outcome was determined at 6 month following trauma by GOS. Confidence of the outcome prediction ranged from 75.8% to 92.1% depending on the logistic regression analysis.

Kaptanglue et al (2001) studied ASDH surgical treatment in a retrospective analysis of 73 cases conducted that GCS at admission is an important prognostic factor and early surgery decreases mortality rate.

Tandon (2001) published acute subdural hematoma as a well entrenched nosological entity, pathologicaly it is usually associated with or, for that matter, secondary to cerebral laceration and contusion. Based on cumulated experience, clinical and pathological studies it is proposed that, for too long the neurosurgeons, “have put emphasis on the clot rather than the totality of the pathological anatomy and that they have focused their therapeutic strategy on removal of the accumulated blood, unmindful of the associated parenchymatous lesion. Not surprisingly, such attempts have been associated with a very high mortality, on the basis of nearly four decades of personal experience and clinical review of the literature, evidence has been provided that to reduce the mortality associated with this condition, it is necessary to evolve a strategy, not only to evacuate the blood but comprehensively deal with the associated parenchymatous lesion and the cascade of secondary insult to the underlying brain”.

Bullock and Chesnut et al (2006) studied surgical management of ASDH. CT thickness of hematoma >10mm and a midline shift of greater than 5mm and patients GCS <9 were regarded as definitive indications for surgery.

MC Nett et al (2007) reviewed the predictive ability of GlasGow Coma Scale in head injured patients.GCS scores are most accurate at predicting the outcome in head injured patients when they are combined with patient age and pupillary response and when broad categories are used. The motor component of the GCS Score yields similar prediction rates as the summed GCS Score and better prediction occurs with very high or low GCS Scores.

Amatol et al (2007) studied prognosis of isolated acute post traumatic SDH in Italy. Pure ASDH without mass lesion patients were analysed based on thickness of hematoma, midline shift, and ASDH volume by CT. Patients outcome was according to the GOS 6 months after the event. They concluded that the presence and size of midline shift was a more important determinant of outcome than ASDH volume or its thickness.

Falerio et al (2008) studied prognostic factors and complication in 89 patients underwent decompressive craniotomy. These patients were analysed over a period of 30 months. Chi square independent test and Fisher test were used to identify the prognostic factors. They concluded admission GCS was a statistically significant predictor of outcome p=0.0309.

Taussky et al(2008) evaluated outcome after acute traumatic subdural and epidural hematoma in Switzerland, a single centre experience concluded that mortality was 41% and Factors such as age, initial GCS and pupil abnormalities still appear to be the most important factors correlating the outcome.

# MATERIALS AND METHODS

This study was a prospective study conducted during the period between the Jan 2007 to Dec 2007 at Madras Institute of Neurology, Government General Hospital, Chennai.

**INCLUSION CRITERIA:**

Inclusion criteria for this study includes

1) Patients having Traumatic acute subdural hematoma admitted at Trauma ward in the year 2007.

2) Only Patients who were treated surgically were included.

**EXCLUSION CRITERIA:**

1) Patients with acute SDH of non-traumatic cause.

2) Thin subdural hematoma managed conservatively.

3) Patients with acute subdural hematoma who are not operated due to other reasons.

Details of the Patients included in the study are entered in the proforma as shown below:

# PROFORMA FOR STUDY

**Prognostic Factors in acute subdural hematoma**

PATIENT DATA:

Name: Age: Sex: I.P. No: MIN. No:

D.O.A: D.O.S: D.O.D:

CLINICAL DATA:

HISTORY

Mode of Injury:

Date and time of injury:

Place of injury:

Mode of transportation:

Date and time of admission:

CLINICAL FINDING:

Glasgow Coma Scale on admission: 3 to 15

Ocular signs : Pupils size and light reflex

Oculo Cephalic Reflex

Neurological focal signs:

Vital signs:

External injury:

Other injuries:

RADIOLOGICAL DATA:

Skull x-ray:

C.T. Scan finding:

Maximum thickness of subdural hematoma:

Midline shift in mm:

State of basal cisterns:

Other parenchymal injuries:

MANAGEMENT:

Date and time of surgery:

Procedure: Craniotomy and Evacuation of subdural hematoma.

Decompressive craniectomy.

OUT COME:

Survival:  Glasgow out come scale:

Death:

The details of time of injury and time of surgery and clinical details like GCS, Brain stem reflexes, CT Scan finding, Type and time of surgery, Out come were noted. The data gathered and analysed. The impact of various prognostic factors like age, admission glasgow coma scale brain stem reflexes ,CT finding ,time interval between injury and surgery and on the out come were analysed using statistical methods using Pearson Chi square test.

# RESULTS

The total number of patients admitted with acute subdural hematoma in the head injury unit were 353. The number of cases operated were 124. The details 124 patient were given in the chart (vide Appendix).

1. The age distribution of the patient was as follows:

| AGE | NO OF PATIENTS |
|---|---|
| 0-10 | 1 |
| 11-20 | 6 |
| 21-30 | 25 |
| 31-40 | 33 |
| 41-50 | 29 |
| 51-60 | 16 |
| 61-70 | 11 |
| 71-80 | 4 |

The maximum number of patients with ASDH was in the 21 to 50 age group.

2. The GCS distribution patients were as follows:

| GCS | NO OF PATIENTS |
|---|---|
| 3-8 | 74 |
| 9-12 | 34 |
| 13-15 | 16 |

The maximum number of patients with operated ASDH had GCS 3 to 8.

The number of patients with operated ASDH with GCS 13 to 15 were far less.

3. The Ocular sign distribution of patients were as follows:

| **PUILLARY SIZE/OCULOCEPHALIC RESPONSE** | **NO OF PATIENTS** |
|---|---|
| PERL/EOM+ | 13 |
| 3MMSRL/DEM+ | 50 |
| 3MMSRL/DEM IMPAIRED | 18 |
| ASYMETRICALPUPIL/DEM IMPAIRED | 20 |
| B/L 4MM NRL DEM ABSENT | 23 |

PERL: Pupils equally reacting to light

SRL : Sluggishly reacting to light

NRL : Not reacting to light

EOM: Extra ocular movement

DEM: Dolls eye movement

Most of the patients had normal pupillary size with sluggish light reflex and preserved doll's eye movement.

4. The CT scan findings of patients were as follows:

| CT FINDING | NO OF PATIENTS |
|---|---|
| No midline shift/basal cistern seen | 23 |
| Midline shift/basal cistern effaced | 91 |
| No midline shift/basal cistern effaced | 9 |

The maximum number of patients had CT finding with midline shift and effaced basal cisterns.

5. Time of injury to the time of surgery was distributed as follows:

| DURATION OF TIME INTERVAL | NO OF PATIENTS |
|---|---|
| 0-3 HRS | O |
| 3-6 HRS | 2 |
| 6-12 HRS | 94 |
| 12-24 HRS | 17 |
| MORE THAN 24 HRS | 11 |

There were no patients presenting within 3 hours of injury.

The majority of patients with ASDH presented for surgery between 6 to 12 hrs.

## CORRELATION BETWEEN VARIOUS FACTORS AND OUTCOME:

### *1. Effect of age on outcome:*

| **Age** | **Outcome** | | | | **Pearson Chi square test** |
|---|---|---|---|---|---|
| | **Died** | | **Alive** | | |
| | **n** | **%** | **n** | **%** | |
| 0-20 | 6 | 85.7 % | 1 | 14.3% | $X^2$= 9.72 |
| 21-40 | 36 | 62.1 % | 22 | 37.9% | P= 0.02 **significant** |
| 41-60 | 39 | 86.7 % | 6 | 13.3% | |
| 60+ | 12 | 85.7 % | 2 | 14.3% | |
| Table total | 93 | 75.0 % | 31 | 25% | |

There was good survival percent in the age group of 21-40. Outcome was poor in the age group above 60 years. The sample size under the age of 20 years is too small to comment on outcome.

***2. Correlation between admission GCS and outcome:***

| GCS | OUT COME | | | | PEARSON CHISQUARE TEST |
|---|---|---|---|---|---|
| | DIED | | ALIVE | | |
| | N | % | N | % | |
| 3-8 | 63 | 85.1% | 11 | 14.9% | X2=20.42<br>P=0.001<br>**SIGNIFICANT** |
| 9-12 | 25 | 73.5% | 9 | 26.5% | |
| 13-15 | 5 | 31.3% | 11 | 68.8% | |
| TABLE TOTAL | 93 | 75% | 31 | 25% | |

The percentage of survival of patients is 25%.

Patients with GCS 13-15 had better outcome

Patients with GCS 3-8 had poor outcome.

The admission GCS had a very positive effect on the outcome with P = 0.001.

***3. Correlation between Pupillary response, Doll's eye movement and outcome:***

| **Pupillary response** | **Out come** | | | | **Pearson chi square test** |
|---|---|---|---|---|---|
| | **Died** | | **Alive** | | |
| | **n** | **%** | **n** | **%** | |
| PERL/DEM | 5 | 35.7% | 9 | 64.3% | $\chi^2$= 17.10 P= 0.002 significant |
| 3MM SRL/DEM+ | 32 | 71.1% | 13 | 28.9% | |
| 3MM SRL/DEMIMPAIRED | 15 | 83.3% | 3 | 16.7% | |
| ASSYMETRY DEMIMPAIRED | 17 | 81.0% | 4 | 19.0% | |
| 4MM NRL DEMIMPAIRED/ ABSENT | 24 | 92.3% | 2 | 7.7% | |
| TABLE TOTAL | 93 | 75.0% | 31 | 25.0% | |

Preserved pupillary light reflex and doll's eye movement correlated with good outcome. Impaired or absent doll's eye movement with impaired or absent light response correlated with poor outcome.

***4. CT scan finding and outcome:***

| CT finding | Outcome | | | | Pearson Chi square test |
|---|---|---|---|---|---|
| | Died | | Alive | | |
| | n | % | n | % | |
| 1 | 15 | 68.2% | 7 | 31.8% | |
| 2 | 72 | 77.4% | 21 | 22.6% | $X^2$= 1.67 |
| 3 | 6 | 66.7% | 3 | 33.3% | P= 0.56 **Not significant** |
| Table total | 93 | 75.0% | 31 | 25.0% | |

NO MIDLINE SHIFT, BASAL CISTERN SEEN = 1

MID LINE SHIFT/BASAL CISTERN EFFACED = 2

NO MIDLINE SHIFT, BASAL CISTERN EFFACED = 3

The degree of mass effect and the state of basal cisterns did not seem to have significant impact on the outcome in the present study.

### *5. Time interval between injury to surgery and outcome:*

| Time (hours) | Outcome | | | | Pearson Chi square test |
|---|---|---|---|---|---|
| | Died | | Alive | | |
| | n | % | n | % | |
| 3-6 | 2 | 100 % | 0 | 0% | $X^2$= 0.89 |
| 6-12 | 71 | 75.5 % | 23 | 24.5% | P= 0.83 **Not significant** |
| 12-24 | 12 | 70.6 % | 5 | 29.4% | |
| >24 | 8 | 72.7% | 3 | 27.3% | |
| Table total | 93 | 75.0 % | 31 | 25.0% | |

The time interval between injury and surgery had no significant impact on outcome in this study.

# DISCUSSION

ASDH is a very major contributor to mortality after head injury. The mortality rate ranges from 40 to 90% in various series. The high percentage of mortality is usually related to the degree of focal and diffuse brain damage associated with the ASDH. Various other factors contribute to morbidity and mortality. The most important of these factors which have been studied include age, GCS at admission, pupillary and brainstem reflexes, degree of mass effect and the state of basal cisterns on CT scan, time between injury to surgery. Many studies have concluded that the most of the above factors affect the outcome in ASDH.

The age of the patient had a significant effect on the outcome survival of patients operated for ASDH. This study shows age between 21-40 has good outcome survival than the children and old age group. The percentage survival was 37.9% in age group of 21-40.The other age groups 0-20, 41-60 and 60+ had more less equally distributed outcome survival of 13%-15%. Pearson chi square test showed x2 = 9.72 with P = 0.02 which is significant. Age has been found to be a very important factor in determining the outcome and by many other authors (Wilberger et al 1991, Kotwica and Brzeziński 1993, Cook et al 1996, Taussky et al 2008).

The admission GCS is a good indicator on the outcome survival of patients operated for acute subdural hematoma in this study. GCS between 3-8 has poor survival rate of 14.9% and most of the patients falls in this GCS group. Patients with GCS 9-12 have moderate survival rate of 26.5%.Patients admitted with GCS 13-15 who are less in number have best survival of 68.8%.The admission GCS has an outcome of P=0.001 which is significant. Thus GCS had more important prognostic value in determining the outcome as suggested by the many authors (Wilberger et al 1991,Kotwica & Brzezinski et al 1993, Massoro et al1996, Falerio et al 2008,Bullock and Chesnut et al 2006,Kaptanglu et al 2001,Yamura et al 2001, Dent et al 1995, Taussky et al 2008).

The pupillary signs has a significant outcome on the survival of patients operated for ASDH. In this study patients with pupils equally reacting to light with dolls eye movement present were less in number but has a survival of 64.3%. Patients admitted moderate in number with pupils 4mmNRL and DEM IMPAIRED or ABSENT has poor survival rate of 7.7% . Pupills 3mm SRL/DEM has most number of patients with moderate survival rate of 28.9 %. Patients with Pupills 3mm and DEM impaired as well as patients with Pupillary asymmetry and DEM impaired has moderate survival rate of 16.7% and 19% respectively.

Pearson chi square test was x2=17.10 and P = 0.002 which is significant. There are other studies supporting that initial pupillary response and eye movement had significant influence on the prognosis as suggested by many authors (Koc et al 1997, Yamaura et al 2001, Kayseri et al1997, Taussky et al 2008).

In this study the CT scan finding of presence or absence of midline shift and basal cistern status had no strong indication for determining the prognosis of ASDH. Most number of patients were admitted with the CT findings of midline shift and basal cistern effacement. The percentage survival varies between 22%-33% in the categories of no midline shift/normal basal cistern, midline shift/basal cistern effacement and no midline shift/ basal cistern effacement, hence it statistically did not influence the prognosis of the patients operated for ASDH. This has been suggested by other authors like Cook et al 1996. Some studies show that CT Scan finding had significant influence on the out come by authors ( Massoro et al1996, Amatol et al 2007, Bullock and Chesnut et al 2006).

The time interval of 6-12hrs between injury and surgery had most number of patients of 94 and the survival percentage was only 24.5%. The other time intervals were 12-24 hrs,> 24 hours and 3-6 hours which had less no of patients and more or less same percentage of survival.

Hence it is concluded that time interval between the injury and surgery has no significant influence on the prognosis of patients operated for acute subdural hematoma statistically. The time interval between injury and surgery was not significant in determining the out come in patients with ASDH as suggested by many authors (Wilberger et al 1991, Kotwica & Brzezinski 1993, Massoro et al 1996, Cook et al 1996). There were no patients presenting for surgery within 3 hours of injury. This is probably because of the inevitable delay in transport of the victims from the site of accident and also because most accidents took place far away from the heart of the city where the hospital is situated.

# CONCLUSION

124 cases of ASDH, which were treated surgically during the year 2007 at the Head Injury Service, Institute of Neurology, Government General Hospital, Chennai, have been studied with regard to the factors determining survival and the following conclusion have been drawn from the study:

1) The overall mortality was 75% (93 cases), which conforms to the other major series.

2) Age, admission GCS, Pupillary and oculo-cephalic responses only had significant impact on survival in ASDH.

3) Time interval between injury and surgery and the CT findings namely, the degree of midline shift and state of the basal cisterns did not affect the survival in the present series.

The limitations of this study are the small number of patients, lack of long term follow up to determine the quality of survival. The number of patients in pediatric and adolescent age group also has been very small to study the outcome in that age group. ASDH continues to pose formidable challenge despite continuing efforts to reduce morbidity and mortality.

# MASTER CHART

| *SRNO* | *AGE* | *SEX* | *GCS* | *TIME INTER hr:mi* | *BRAINSTEM REFLEX* | *CTSCAN FINDING* | *OUTCOME* |
|---|---|---|---|---|---|---|---|
| 1 | 55 | M | 11 | 12:30 | 3mmsrldem+ | nomls/bc+ | EXP |
| 2 | 65 | M | 14 | <12 | 3mmsrldem+ | Mlshift/bc- | EXP |
| 3 | 22 | M | 9 | 11 | 3mmsrldem+ | Mlshift/bc- | EXP |
| 4 | 40 | M | 9 | 11:30 | 3mmsrldem+ | Mlshift/bc- | EXP |
| 5 | 25 | M | 12 | 15 | 3mmsrldem+ | Nomls/bc+ | E4VIM5RTPAUCITY |
| 6 | 42 | M | 7 | <12 | 4mmnrldem- | Mlshift/bc- | EXP |
| 7 | 50 | M | 11 | 10 | 3mmsrldem+ | Mlshift/bc+ | EXP |
| 8 | 39 | M | 4 | <12 | 4mmnrldem- | Mlshift/bc- | EXP |
| 9 | 40 | F | 14 | 26 | 3mmsrleom+ | Nomls/bc+ | EXP |
| 10 | 40 | F | 8 | 18 | 3mmsrldem+ | Mlshift/bc- | EXP |
| 11 | 33 | M | 5 | 18:30 | Lt3mmdem+ | Nomls/bc+ | E4V3M6RTGL OBEINJURY |
| 12 | 38 | M | 12 | <12 | Rt3mmdem+ | Mlshift/b | E4V4M6 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | | | | | c- | |
| 13 | 37 | M | 5 | 6:45 | 3mmsrldem+ | Nomls/bc- | EXP |
| 14 | 52 | M | 8 | 8 | 3mmsrldem- | Mlshift/bc- | EXP |
| 15 | 35 | M | 5 | <12 | 4mmnrldem- | Nomls/bc- | E4V5M6 |
| 16 | 60 | M | 12 | <12 | 3mmsrldem+ | Nomls/bc+ | EXP |
| 17 | 60 | F | 5 | 24 | 4mmnrldem- | Mlshift/bc- | EXP |
| 18 | 58 | M | 3 | <12 | 4mmnrldem- | Mlshift/bc- | EXP |
| 19 | 35 | M | 8 | 19:30 | Rt3mmlt4mmsrldem+ | Nomls/bc- | EXP |
| 20 | 40 | M | 10 | <12 | 3mmsrldem+ | Nomls/bc- | E4V4M5 |

| ***SRno*** | ***AGE*** | ***SEX*** | ***GCS*** | ***TIME hr:mi*** | ***BRAIN STEM REFLEX*** | ***CT SCAN FINDING*** | ***OUTCOME*** |
|---|---|---|---|---|---|---|---|
| 21 | 36 | M | 10 | 43:30 | 3mmsrldem+ | Nomls/bc- | EXP |
| 22 | 49 | M | 3 | 11 | 4mmnrldem- | Mlshift/bc- | EXP |
| 23 | 12 | M | 13 | <12 | Rt4mmnrllt 3mmsrldem- | Nomls/bc+ | 15 LT PAUCITY |
| 24 | 40 | M | 5 | 4DAYS | Lt2mmrt 3mmsrldem- | Mlshift/bc- | EXP |
| 25 | 80 | M | 9 | 12-24 | 3mmsrldem- | Mlshift/bc- | EXP |
| 26 | 45 | M | 8 | <12 | 3mmsrldem- | Nomls/bc+ | EXP |
| 27 | 45 | M | 8 | <12 | 3mmsrldem+ | Mlshift/bc- | EXP |
| 28 | 43 | M | 3 | <12 | 3mmsrldem+ | Mlshift/bc- | EXP |

| *SRno* | *AGE* | *SEX* | *GCS* | *TIME hr:mi* | *BRAIN STEM REFLEX* | *CT SCAN FINDING* | *OUTCOME* |
|---|---|---|---|---|---|---|---|
| 29 | 65 | M | 6 | 9 | Rt3mmsrllt 5mmnrldem- | Mlshift/bc- | EXP |
| 30 | 65 | M | 3 | <24 | Rt4mmnrllt 3mmsrldem- | Mlshift/bc- | EXP |
| 31 | 35 | M | 5 | 26 | 4mmnrldem- | Mlshift/bc- | EXP |
| 32 | 52 | M | 13 | <12 | 3mmsrldem- | Mlshift/bc- | 15 LT PAUCITY |
| 33 | 65 | M | 10 | 18:30 | Rt4mmnrllt 3mmsrldem- | Nomls/bc+ | EXP |
| 34 | 25 | M | 4 | 8:30 | 4mmnrldem- | Mlshift/bc- | EXP |
| 35 | 59 | M | 5 | <12 | 4mmnrldem- | Mlshift/bc- | EXP |
| 36 | 38 | M | 9 | 5:45 | 3mmsrldem+ | Mlshift/bc- | EXP |
| 37 | 62 | M | 14 | 38:30 | Perl eom+ | Mlshift/bc- | 15 |
| 38 | 35 | M | 4 | <12 | 3mmsrldem+ | Mlshift/bc- | 14 LT KERRATITIS |
| 39 | 20 | M | 7 | <12 | 3mmsrldem+ | Mlshift/bc- | EXP |
| 40 | 35 | M | 5 | <12 | 3mmsrldem+ | Mlshift/bc- | 14 LT $7^{TH}$ PALSY |

| *SRNO* | *AGE* | *SEX* | *GCS* | *TIME INTERVAL hrs:min* | *BRAINSTEM REFEFLEX* | *CTfinding* | *OUTCOME* |
|---|---|---|---|---|---|---|---|
| 41 | 44 | M | 14 | 6:45 | Perl eom+ | Mlshift/bc- | 15 |
| 42 | 20 | M | 9 | <12 | 3mmsrldem+ | Mlshift/bc- | EXP |
| 43 | 28 | M | 8 | 9 | 3mmsrldem+ | Mlshift/bc- | EXP |
| 44 | 26 | M | 5 | 17 | Rt3mmsrllt 5mmnrldem- | Mlshift/bc- | EXP |
| 45 | 32 | F | 13 | <12 | Perl eom+ | Mlshift/bc- | 15 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 46 | 23 | M | 9 | 42:30 | Perl eom+ | Mlshift/bc- | EXP |
| 47 | 65 | F | 12 | 18 | 4mmnrldem+ | Nomls/bc+ | EXP |
| 48 | 47 | F | 11 | 17 | Perl eom+ | Mlshift/bc- | EXP |
| 49 | 30 | M | 12 | <12 | Perl eom+ | Nomls/bc+ | 15 |
| 50 | 45 | F | 11 | 7:30 | 3mmsrldem+ | Mlshift/bc- | EXP |
| 51 | 60 | F | 9 | 9 | Perl eom+ | Mlshift/bc- | EXP |
| 52 | 30 | M | 12 | 3DAY4hr | 3mmsrldem+ | Mlshift/bc- | EXP |
| 53 | 40 | M | 9 | <12 | Perl eom+ | Mlshift/bc- | E4V3M5 |
| 54 | 65 | M | 14 | 18 | Perl eom+ | Mlshift/bc- | 15 |
| 55 | 60 | M | 9 | <12 | 3mmsrldem- | Mlshift/bc- | EXP |
| 56 | 31 | M | 8 | <12 | 3mmsrldem- | Mlshift/bc- | EXP |
| 57 | 40 | M | 7 | <12 | 4mmnrldem- | Mlshift/bc- | EXP |
| 58 | 46 | M | 7 | <12 | 4mmnrldem- | Mlshift/bc- | EXP |
| 59 | 50 | M | 5 | 11 | 4mmnrldem- | Mlshift/bc- | EXP |
| 60 | 42 | M | 3 | 14:30 | Rt4mmnrllt 3mmsrldem- | Mlshift/bc- | EXP |

| ***SRNO*** | ***AGE*** | ***SEX*** | ***GCS*** | ***TIME INTERVAL hr:min*** | ***BRAINSTEMREFLEX*** | ***CT FINDING*** | ***OUTCOME*** |
|---|---|---|---|---|---|---|---|
| 61 | 50 | M | 6 | <12 | 4mmnrldem- | Mlshift/bc- | EXP |
| 62 | 35 | M | 3 | <12 | 4mmnrldem- | Mlshift/bc- | EXP |
| 63 | 77 | M | 8 | <12 | 3mmdem+ | Mlshift/bc- | EXP |
| 64 | 43 | M | 5 | <12 | 3mmdem- | Mlshift/bc- | EXP |
| 65 | 24 | M | 11 | 54 | Perl eom+ | Mlshift/bc- | E4V3M6 |
| 66 | 32 | M | 8 | <12 | Rt3mmlt5mmsrldem+ | Mlshift/bc- | 15 |
| 67 | 42 | M | 8 | 10:30 | 4mmnrldem- | Mlshift/bc- | 15 |
| 68 | 40 | M | 10 | 48 | Perl eom+ | Nomls/bc+ | EXP |
| 69 | 45 | M | 4 | 15 | Rt3mmlt4mmnrldem- | Mlshift/bc- | EXP |
| 70 | 27 | M | 12 | <12 | 3mmdem+ | Mlshift/bc- | EXP |
| 71 | 38 | M | 15 | <12 | Perl eom+ | Mlshift/bc- | 15 |
| 72 | 38 | M | 4 | <12 | Rt5mmlt4mmnrldem- | Mlshift/bc- | EXP |
| 73 | 45 | M | 10 | <12 | 3mmdem+ | Nomls/b | EXP |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | | | | | c+ | |
| 74 | 65 | M | 3 | <12 | 4mmnrldem- | Nomls/bc+ | EXP |
| 75 | 40 | M | 4 | 5 | Lt4mmnrlrt3mmsrldem- | Mlshift/bc- | EXP |
| 76 | 70 | F | 13 | 8 | 3mmdem+ | Mlshift/bc- | EXP |
| 77 | 25 | M | 7 | 6:30 | 3mmdem+ | Mlshift/bc- | EXP |
| 78 | 45 | M | 5 | 6:05 | Rt2mmsrllt4mmnrldem- | Mlshift/bc- | EXP |
| 79 | 25 | M | 7 | 40 | Rt4mmsrllt3mmrldem+ | Mlshift/bc- | 15 |
| 80 | 33 | M | 5 | <12 | 3mmdem+ | Nomls/bc+ | E4V3M6 |

| ***SRNO*** | ***AGE*** | ***SEX*** | ***GCS*** | ***TIME INTER hrs:min*** | ***BRAIN STEM REFLEX*** | ***CT FINDING*** | ***OUTCOME*** |
|---|---|---|---|---|---|---|---|
| 81 | 47 | M | 4 | <12 | 4mmnrldem- | Mlshift/bc- | EXP |
| 82 | 48 | F | 5 | 11 | Rt3mmnrllt2mmnrldem- | Nomls/bc- | EXP |
| 83 | 35 | M | 3 | <12 | 4mmnrldem- | Nomls/bc+ | EXP |
| 84 | 8/12 | M | 3 | 10:15 | 4mmnrldem- | Nomls/bc- | EXP |
| 85 | 27 | M | 4 | 7 | Rt5mmnrllt3mmsrlde | Nomls/bc | EXP |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | | | | m- | - | |
| 86 | 55 | F | 7 | <12 | Rt3mmlt5mmnrldem- | Mlshift/bc- | EXP |
| 87 | 54 | F | 5 | <12 | 3mmsrldem- | Nomls/bc+ | EXP |
| 88 | 42 | M | 8 | 8DAYS | 3mmsrldem- | Mlshift/bc- | EXP |
| 89 | 40 | M | 3 | 8 | 4mmnrldem- | Mlshift/bc- | EXP |
| 90 | 64 | M | 9 | 8:30 | 3mmsrldem+ | Mlshift/bc- | EXP |
| 91 | 60 | M | 3 | <12 | 4mmnrldem- | Mlshift/bc- | EXP |
| 92 | 58 | M | 4 | <12 | 4mmnrldem- | Mlshift/bc- | EXP |
| 93 | 50 | M | 11 | <12 | 3mmsrldem- | Mlshift/bc- | EXP |
| 94 | 20 | M | 4 | 11:30 | 5mmnrldem- | Mlshift/bc- | EXP |
| 95 | 21 | M | 9 | <12 | 3mmsrldem- | Mlshift/bc- | EXP |
| 96 | 30 | M | 5 | <12 | 3mmsrldem- | Mlshift/bc- | EXP |
| 97 | 24 | M | 14 | 8 | 3mmsrldem+ | Mlshift/bc- | 15 |
| 98 | 50 | F | 4 | 10:30 | 3mmsrldem- | Mlshift/bc- | EXP |
| 99 | 25 | M | 7 | 6:30 | 3mmsrldem- | Mlshift/bc- | EXP |
| 100 | 26 | M | 4 | 8 | 5mmnrldem- | Mlshift/bc- | EXP |

| ***SRNO*** | ***AGE*** | ***SEX*** | ***GCS*** | ***TIME INTERVAL hrs:min*** | ***Brainstem reflex*** | ***CT finding*** | ***OUTCOME*** |
|---|---|---|---|---|---|---|---|
| 101 | 50 | F | 13 | 7 | 3mmsrldem+ | Mlshift/bc- | EXP |
| 102 | 41 | M | 6 | 28 | rt3mmnrllt2mmsrldem- | Mlshift/bc- | EXP |
| 103 | 13 | M | 7 | <12 | 3mmsrldem- | Mlshift/bc- | EXP |
| 104 | 40 | M | 5 | 8:20 | Rt4mmlt5mmnrldem- | Mlshift/bc- | EXP |
| 105 | 40 | M | 7 | 10 | Rt3mmsrllt2mmsrldem- | Mlshift/bc- | EXP |
| 106 | 66 | M | 3 | <12 | 4mmnrldem- | Mlshift/bc- | EXP |
| 107 | 35 | M | 5 | 6:10 | 3mmsrldem- | Mlshift/bc- | E4V3M6 |
| 108 | 75 | M | 10 | 12 | 3mmsrldem+ | Mlshift/bc- | EXP |
| 109 | 25 | M | 7 | <12 | 3mmsrldem- | Mlshift/bc- | EXP |
| 110 | 35 | M | 9 | <12 | 3mmsrldem+ | Nomls/bc+ | EXP |
| 111 | 28 | M | 15 | 10:15 | 3mmsrldem+ | Mlshift/bc- | 15 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 112 | 45 | M | 9 | 19 | 3mmsrldem+ | Mlshift/bc- | E4V3M6 |
| 113 | 24 | M | 13 | 7:30 | 3mmsrldem+ | Mlshift/bc- | 15 |
| 114 | 35 | M | 4 | <12 | 3mmsrldem+ | Mlshift/bc- | EXP |
| 115 | 45 | M | 15 | 10 | 3mmsrldem+ | Mlshift/bc- | EXP |
| 116 | 58 | M | 8 | <12 | 3mmsrldem+ | Mlshift/bc- | EXP |
| 117 | 60 | M | 9 | <12 | 3mmsrldem+ | Mlshift/bc- | EXP |
| 118 | 26 | M | 9 | 11 | 3mmsrldem- | Mlshift/bc- | E4V1M6 |
| 119 | 52 | M | 5 | <12 | 3mmsrldem+ | Nomls/bc+ | E4V5M6 |
| 120 | 25 | M | 13 | 12:30 | 3mmsrldem+ | Nomls/bc+ | 15 |

| ***SRNO*** | ***AGE*** | ***SEX*** | ***GCS*** | ***TIME INTERVAL hrs:min*** | ***brainstemreflex*** | ***CT finding*** | ***OUTCOME*** |
|---|---|---|---|---|---|---|---|
| 121 | 27 | M | 4 | <12 | 3mmsrldem+ | Mlshift/bc- | EXP |
| 122 | 45 | M | 3 | <12 | 3mmsrldem+ | Nomls/bc+ | EXP |
| 123 | 23 | M | 5 | 8 | Rt4mmnrllt | Nomls/bc | E4V4M5 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | | | | 3mmsrldem- | - | |
| 124 | 43 | M | 11 | 8 | 3mmsrldem+ | Mlshift/bc<br>- | E4V3M5 |

# NUMERICAL ABBREVIATION

| |
|---|
| BRAINSTEM REFLEXES |
| PERL/DEM+ =No.1 |
| 3 MM SRL/DEM+ =No.2 |
| 3MM SRL/DEMIMPAIRED=No.3 |
| ASSYMETRY DEMIMPAIRED=No.4 |
| 4MM NRL DEM IMPAIRED/ ABSENT=No.5 |
| CT SCAN |
| NO MIDLINE SHIFT BASAL CISTERN SEEN=No.1 |
| MID LINE SHIFT/BASAL CISTERN EFFACED=No.2 |
| NOMIDLINE SHIF BASALCISTERNEFFACED=No.3 |

**ABBREVIATION**

NO MIDLINESHIFT/BASALCISTERN SEEN=NOmlshift/bc+

MIDLINESHIFT/BASALCISTERN EFFACED=mlshift/bc-

NOMIDLINESHIFT/BASALCISTERN EFFACED=nomls/bc-

PERL= PUPILS EQUALLY REACTING TO LIGHT

EOM=EXTRAOCULAR MOVEMENT

DEM=DOLLS EYE MOVEMENT

Srl = sluggishly reacting to light

Rl= reacting to light

Nrl=not reacting to light

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| S.No | GCS | Dea/Ali | Time | Dea/Ali | CT | Dea/Ali | Brain Stem | Dea/Ali | Age | Dea/Ali |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 2 | 0 | 4 | 0 | 1 | 0 | 2 | 0 | 6 | 0 |
| 2 | 3 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 7 | 0 |
| 3 | 2 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 3 | 0 |
| 4 | 2 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 4 | 0 |
| 5 | 2 | 1 | 4 | 1 | 1 | 1 | 2 | 1 | 3 | 1 |
| 6 | 1 | 0 | 3 | 0 | 2 | 0 | 5 | 0 | 5 | 0 |
| 7 | 2 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 5 | 0 |
| 8 | 1 | 0 | 3 | 0 | 2 | 0 | 5 | 0 | 4 | 0 |
| 9 | 3 | 0 | 5 | 0 | 1 | 0 | 2 | 0 | 4 | 0 |
| 10 | 1 | 0 | 4 | 0 | 2 | 0 | 2 | 0 | 4 | 0 |
| 11 | 1 | 1 | 4 | 1 | 1 | 1 | 2 | 1 | 4 | 1 |
| 12 | 2 | 1 | 3 | 1 | 2 | 1 | 2 | 1 | 4 | 1 |
| 13 | 1 | 0 | 3 | 0 | 3 | 0 | 2 | 0 | 4 | 0 |
| 14 | 1 | 0 | 3 | 0 | 2 | 0 | 3 | 0 | 6 | 0 |
| 15 | 1 | 1 | 3 | 1 | 3 | 1 | 5 | 1 | 4 | 1 |
| 16 | 2 | 0 | 3 | 0 | 1 | 0 | 2 | 0 | 6 | 0 |
| 17 | 1 | 0 | 4 | 0 | 2 | 0 | 5 | 0 | 6 | 0 |
| 18 | 1 | 0 | 3 | 0 | 2 | 0 | 5 | 0 | 6 | 0 |

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|---|---|---|---|---|---|---|---|---|---|---|
| 19 | 1 | 0 | 4 | 0 | 3 | 0 | 4 | 0 | 2 | 0 |
| 20 | 2 | 1 | 3 | 1 | 3 | 1 | 2 | 1 | 4 | 1 |
| 21 | 2 | 0 | 5 | 0 | 3 | 0 | 2 | 0 | 4 | 0 |
| 22 | 1 | 0 | 3 | 0 | 2 | 0 | 5 | 0 | 5 | 0 |
| 23 | 3 | 1 | 3 | 1 | 1 | 1 | 4 | 1 | 2 | 1 |
| 24 | 1 | 0 | 5 | 0 | 2 | 0 | 4 | 0 | 4 | 0 |
| 25 | 2 | 0 | 4 | 0 | 2 | 0 | 3 | 0 | 8 | 0 |
| 26 | 1 | 0 | 3 | 0 | 1 | 0 | 3 | 0 | 5 | 0 |
| 27 | 1 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 5 | 0 |
| 28 | 1 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 5 | 0 |
| 29 | 1 | 0 | 3 | 0 | 2 | 0 | 4 | 0 | 7 | 0 |
| 30 | 1 | 0 | 4 | 0 | 1 | 0 | 4 | 0 | 7 | 0 |
| 31 | 1 | 0 | 5 | 0 | 2 | 0 | 5 | 0 | 4 | 0 |
| 32 | 3 | 1 | 3 | 1 | 2 | 1 | 3 | 1 | 6 | 1 |
| 33 | 2 | 0 | 4 | 0 | 1 | 0 | 4 | 0 | 7 | 0 |
| 34 | 1 | 0 | 3 | 0 | 2 | 0 | 5 | 0 | 3 | 0 |
| 35 | 1 | 0 | 3 | 0 | 2 | 0 | 5 | 0 | 6 | 0 |
| 36 | 2 | 0 | 2 | 0 | 2 | 0 | 2 | 0 | 4 | 0 |
| 37 | 3 | 1 | 5 | 1 | 2 | 1 | 1 | 1 | 7 | 1 |
| 38 | 1 | 1 | 3 | 1 | 2 | 1 | 2 | 1 | 4 | 1 |
| 39 | 1 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 2 | 0 |

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|---|---|---|---|---|---|---|---|---|---|---|
| 40 | 1 | 1 | 3 | 1 | 2 | 1 | 2 | 1 | 4 | 1 |
| 41 | 3 | 1 | 3 | 1 | 2 | 1 | 1 | 1 | 5 | 1 |
| 42 | 2 | 0 | 3 | 0 | 1 | 0 | 2 | 0 | 2 | 0 |
| 43 | 1 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 3 | 0 |
| 44 | 1 | 0 | 4 | 0 | 2 | 0 | 4 | 0 | 3 | 0 |
| 45 | 3 | 1 | 3 | 1 | 2 | 1 | 1 | 1 | 4 | 1 |
| 46 | 2 | 0 | 5 | 0 | 2 | 0 | 1 | 0 | 3 | 0 |
| 47 | 2 | 0 | 4 | 0 | 1 | 0 | 5 | 0 | 7 | 0 |
| 48 | 2 | 0 | 4 | 0 | 2 | 0 | 1 | 0 | 5 | 0 |
| 49 | 2 | 1 | 3 | 1 | 1 | 1 | 1 | 1 | 3 | 1 |
| 50 | 2 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 5 | 0 |
| 51 | 2 | 0 | 3 | 0 | 2 | 0 | 1 | 0 | 6 | 0 |
| 52 | 2 | 0 | 5 | 0 | 2 | 0 | 2 | 0 | 3 | 0 |
| 53 | 2 | 1 | 3 | 1 | 2 | 1 | 1 | 1 | 4 | 1 |
| 54 | 3 | 1 | 4 | 1 | 2 | 1 | 1 | 1 | 7 | 1 |
| 55 | 2 | 0 | 3 | 0 | 2 | 0 | 3 | 0 | 6 | 0 |
| 56 | 1 | 0 | 3 | 0 | 2 | 0 | 3 | 0 | 4 | 0 |
| 57 | 1 | 0 | 3 | 0 | 2 | 0 | 5 | 0 | 4 | 0 |
| 58 | 1 | 0 | 3 | 0 | 2 | 0 | 5 | 0 | 5 | 0 |
| 59 | 1 | 0 | 3 | 0 | 2 | 0 | 5 | 0 | 5 | 0 |
| 60 | 1 | 0 | 4 | 0 | 2 | 0 | 4 | 0 | 5 | 0 |

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|---|---|---|---|---|---|---|---|---|---|---|
| 61 | 1 | 0 | 3 | 0 | 2 | 0 | 5 | 0 | 5 | 0 |
| 62 | 1 | 0 | 3 | 0 | 2 | 0 | 5 | 0 | 4 | 0 |
| 63 | 1 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 8 | 0 |
| 64 | 1 | 0 | 3 | 0 | 2 | 0 | 3 | 0 | 5 | 0 |
| 65 | 2 | 1 | 5 | 1 | 2 | 1 | 1 | 1 | 3 | 1 |
| 66 | 1 | 1 | 3 | 1 | 2 | 1 | 4 | 1 | 4 | 1 |
| 67 | 1 | 1 | 3 | 1 | 2 | 1 | 5 | 1 | 5 | 1 |
| 68 | 2 | 0 | 5 | 0 | 1 | 0 | 1 | 0 | 4 | 0 |
| 69 | 1 | 0 | 4 | 0 | 2 | 0 | 4 | 0 | 5 | 0 |
| 70 | 2 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 3 | 0 |
| 71 | 3 | 1 | 3 | 1 | 2 | 1 | 1 | 1 | 4 | 1 |
| 72 | 1 | 0 | 3 | 0 | 2 | 0 | 4 | 0 | 4 | 0 |
| 73 | 2 | 0 | 3 | 0 | 1 | 0 | 1 | 0 | 5 | 0 |
| 74 | 1 | 0 | 3 | 0 | 1 | 0 | 5 | 0 | 7 | 0 |
| 75 | 1 | 0 | 2 | 0 | 2 | 0 | 4 | 0 | 4 | 0 |
| 76 | 3 | 0 | 3 | 0 | 1 | 0 | 2 | 0 | 7 | 0 |
| 77 | 1 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 3 | 0 |
| 78 | 1 | 0 | 3 | 0 | 2 | 0 | 4 | 0 | 5 | 0 |
| 79 | 1 | 1 | 5 | 1 | 2 | 1 | 4 | 1 | 3 | 1 |
| 80 | 1 | 1 | 3 | 1 | 1 | 1 | 2 | 1 | 4 | 1 |
| 81 | 1 | 0 | 3 | 0 | 2 | 0 | 5 | 0 | 5 | 0 |

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|---|---|---|---|---|---|---|---|---|---|---|
| 82 | 1 | 0 | 3 | 0 | 3 | 0 | 4 | 0 | 5 | 0 |
| 83 | 1 | 0 | 3 | 0 | 1 | 0 | 5 | 0 | 4 | 0 |
| 84 | 1 | 0 | 3 | 0 | 3 | 0 | 5 | 0 | 1 | 0 |
| 85 | 1 | 0 | 3 | 0 | 3 | 0 | 4 | 0 | 3 | 0 |
| 86 | 1 | 0 | 3 | 0 | 2 | 0 | 4 | 0 | 6 | 0 |
| 87 | 1 | 0 | 3 | 0 | 1 | 0 | 3 | 0 | 6 | 0 |
| 88 | 1 | 0 | 3 | 0 | 2 | 0 | 3 | 0 | 5 | 0 |
| 89 | 1 | 0 | 3 | 0 | 2 | 0 | 5 | 0 | 4 | 0 |
| 90 | 2 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 7 | 0 |
| 91 | 1 | 0 | 3 | 0 | 2 | 0 | 5 | 0 | 6 | 0 |
| 92 | 1 | 0 | 3 | 0 | 2 | 0 | 5 | 0 | 6 | 0 |
| 93 | 2 | 0 | 3 | 0 | 2 | 0 | 3 | 0 | 5 | 0 |
| 94 | 1 | 0 | 3 | 0 | 2 | 0 | 5 | 0 | 2 | 0 |
| 95 | 1 | 0 | 3 | 0 | 2 | 0 | 3 | 0 | 3 | 0 |
| 96 | 1 | 0 | 3 | 0 | 2 | 0 | 3 | 0 | 3 | 0 |
| 97 | 3 | 1 | 3 | 1 | 2 | 1 | 2 | 1 | 3 | 1 |
| 98 | 1 | 0 | 3 | 0 | 2 | 0 | 3 | 0 | 5 | 0 |
| 99 | 1 | 0 | 3 | 0 | 2 | 0 | 3 | 0 | 3 | 0 |
| 100 | 1 | 0 | 3 | 0 | 2 | 0 | 5 | 0 | 3 | 0 |
| 101 | 3 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 5 | 0 |
| 102 | 1 | 0 | 5 | 0 | 2 | 0 | 4 | 0 | 5 | 0 |

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|---|---|---|---|---|---|---|---|---|---|---|
| 103 | 1 | 0 | 3 | 0 | 2 | 0 | 3 | 0 | 2 | 0 |
| 104 | 1 | 0 | 3 | 0 | 2 | 0 | 4 | 0 | 4 | 0 |
| 105 | 1 | 0 | 3 | 0 | 2 | 0 | 4 | 0 | 4 | 0 |
| 106 | 1 | 0 | 3 | 0 | 2 | 0 | 5 | 0 | 7 | 0 |
| 107 | 1 | 1 | 3 | 1 | 2 | 1 | 3 | 1 | 4 | 1 |
| 108 | 2 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 8 | 0 |
| 109 | 1 | 0 | 3 | 0 | 2 | 0 | 3 | 0 | 3 | 0 |
| 110 | 2 | 0 | 3 | 0 | 1 | 0 | 2 | 0 | 4 | 0 |
| 111 | 3 | 1 | 3 | 1 | 2 | 1 | 1 | 1 | 3 | 1 |
| 112 | 2 | 1 | 4 | 1 | 2 | 1 | 2 | 1 | 5 | 1 |
| 113 | 3 | 1 | 3 | 1 | 2 | 1 | 2 | 1 | 3 | 1 |
| 114 | 1 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 4 | 0 |
| 115 | 3 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 5 | 0 |
| 116 | 1 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 6 | 0 |
| 117 | 2 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 6 | 0 |
| 118 | 2 | 1 | 3 | 1 | 2 | 1 | 3 | 1 | 3 | 1 |
| 119 | 1 | 1 | 3 | 1 | 1 | 1 | 2 | 1 | 6 | 1 |
| 120 | 3 | 1 | 4 | 1 | 1 | 1 | 2 | 1 | 3 | 1 |
| 121 | 1 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 3 | 0 |
| 122 | 1 | 0 | 3 | 0 | 2 | 0 | 2 | 0 | 5 | 0 |
| 123 | 1 | 1 | 3 | 1 | 3 | 1 | 4 | 1 | 3 | 1 |

124 2 1 3 1 2 1 2 1 5 1

GLASGOW COMA SCALE

3-8=No.1

9-12=No.2

13-15=No.3

DEATH =0

ALIVE=1

BRAINSTEM REFLEXES

PERL/DEM+ =No.1

3 MM SRL/DEM+ =No.2

3MM SRL/DEMIMPAIRED=No.3

ASSYMETRY DEMIMPAIRED=No.4

4MM NRL DEM IMPAIRED/ ABSENT=No.5

CT SCAN

NO MIDLINE SHIFT BASAL CISTERN SEEN=No.1

MID LINE SHIFT/BASAL CISTERN EFFACED=No.2

NOMIDLINE SHIF BASALCISTERNEFFACED=No.3

TIME OF INJURY AND TREATEMENT

0-3 HOURS=No.1

3-6 HOURS=No.2

6-12 HOURS=No.3

12-24 HOURS=No.4

>24 HOURS=No.5

AGE

0 to 10 YEARS=No.1

11 to 20 YEARS=No.2

21 to 30 YEARS=No.3

31 to 40 YEARS=No.4

41 to 50 YEARS=No.5

51 to 60 YEARS=No.6

61 to 70 YEARS=No.7

71 to 80 YEARS=No.8